

CASE REPORT

REPETITIVE ATRIAL STANDSTILL WITH IDIOVENTRICULAR RHYTHM FROM HYPERKALÆMIA

BY

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The electrocardiographic changes brought about by high serum levels of potassium are well documented. In the more severe cases, both atrial standstill and idioventricular rhythm are known to occur. However, the atrial standstill is usually for one or two beats only. As far as is known, the occurrence of the two together appearing as a repetitive arrhythmia has not previously been recorded.

High levels of serum potassium often occur in acute renal tubular lesions and less frequently in chronic renal disease. In the case reported here the hyperkalæmia seems to have occurred with uræmia from a chronic tubular disorder, without any episode of anuria or oliguria having been noted. Sulphonamides and their derivatives usually cause acute kidney lesions but in this case they led to a chronic chemical nephritis.

Case Report

An Asian merchant, aged 49 years, was admitted to hospital for the investigation of persistent vomiting of one year's duration. He had lost some weight, but there were no other symptoms. A barium meal and follow through and a barium enema had revealed no abnormalities.

On examination he was rather obese and looked ill, but apart from anæmia and an indirect right inguinal hernia there were no abnormal physical findings. The blood pressure varied between 110/70 and 160/90 mm. Hg. The hæmoglobin was 44 per cent (6.5 g./100 ml.) and a blood film showed hypochromic, microcytic red cells. The white blood count and differential were normal. Examination of the stool revealed no microscopic abnormalities and no occult blood: no pathogens were cultured. The urine was acid, contained a trace of albumen, and a few pus cells and red blood cells. These slight deviations from normality were confirmed on two other occasions.

A few days after admission, and while investigations were proceeding, the patient complained of substernal pain and breathlessness. On examination his general condition was worse and his breathing was of the Kussmaul acidotic kind. The blood pressure was 135/70 mm. Hg. The pulse was regular but the rate varied, at times it was 100 a minute and at other times 52 a minute. These two different rates alternated, each being maintained for several minutes and then abruptly changing. The jugular venous pressure was slightly raised. The form of the jugular pulse was carefully studied: during the periods with the rapid rate, both *a* and *v* waves were readily seen, whilst during the slow periods only a single, presumably *v*, wave was seen. No "cannon" waves were seen during the slow periods. It was concluded that periodic atrial standstill with idioventricular rhythm was occurring.

An electrocardiogram (Fig. 1) showed widening of the QRS complex and high, peaked, narrow-based T waves in leads V2, V3, and V4, suggestive of hyperkalæmia. Electrocardiographic study of the arrhythmia (Fig. 2) confirmed that the rapid periods were of normal rhythm and small P waves could be seen: they were clearly not U waves or they would have been present when the rate was slow. The slow periods were of atrial standstill and idioventricular rhythm, no P waves being seen. On one occasion (Fig. 2) atrial activity with irregular ventricular response was observed for a short while. The serum electrolytes were estimated and were as follows: sodium 130 m.eq./l., potassium 8.2 m.eq./l., and calcium 3.8 m.eq./l. The blood urea was 192 mg./100 ml.

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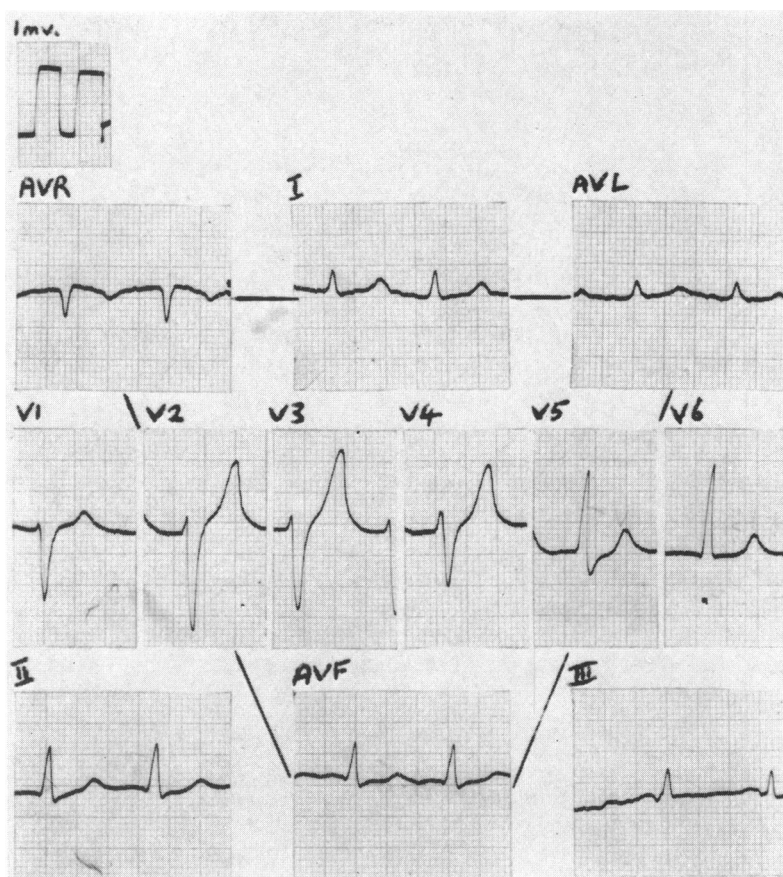


FIG. 1.—Electrocardiogram showing wide QRS complexes and tall, peaked T waves of hyperkalemia in leads V2, V3, and V4.

The repetitive arrhythmia was maintained for about six hours, after which the rate dropped to a constant 40 a minute. The beat was so feeble that the pulse could not be felt at the wrist. This slow rate continued for about half an hour until the patient died. Although no cardiogram was taken at this time, it seems likely that cardiac arrest rather than ventricular fibrillation was the immediate cause of death.

At post-mortem examination the heart had stopped in diastole. It weighed 380 g. The valves were all normal. Slight intimal thickening was seen in the left anterior descending and right coronary arteries. There was slight fatty streaking of the intima just above the aortic valve. The left ventricle was 12 mm. thick, and the right ventricular outflow tract 4 mm. thick. Microscopically the heart lesions were considered to be insignificant. The main coronary arteries showed a moderate degree of sclerosis with intimal thickening; likewise many of the small vessels in the myocardium. There was no evidence of damage to or replacement of the cardiac muscle fibres. There was neither hypertrophy nor cellular infiltration, although there was a slight increase of fibrous tissue around some of the blood vessels, resembling old healed Aschoff bodies.

The kidneys were surrounded by heavy deposits of fat. The capsule was normal and stripped easily. The surfaces were scarred and shrunken. The left kidney weighed 54 g. and the right 86 g. Microscopically they showed a most interesting lesion. The glomeruli were mostly within normal limits, but the tubules were almost completely concealed by gross interstitial fibrosis. Large amounts of crystalline material were present both in the tubules and in the interstitial tissue. The crystals had the typical appearance of sulpho-namide crystals. No chemical identification was undertaken, but by polarized light the crystals were seen to be birefringent.

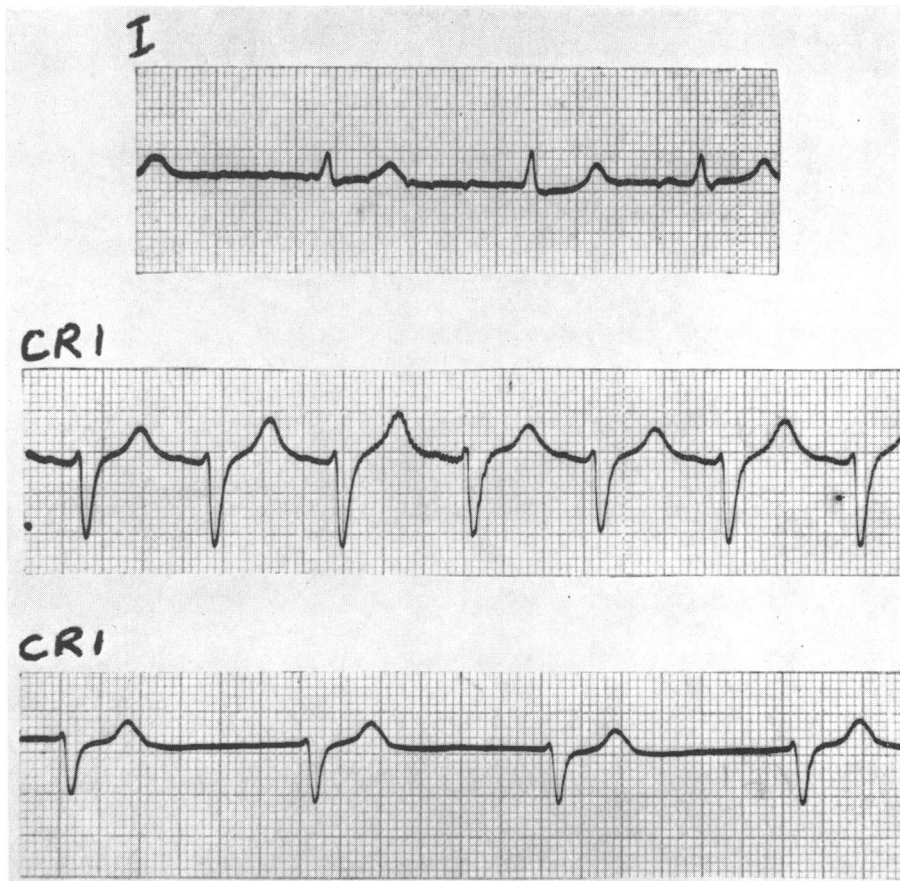


FIG. 2.—Electrocardiogram showing in lead I rapid atrial beats with irregular ventricular response. Lead CR1 upper tracing, shows normal rhythm with small but clear P waves, and lower tracing, atrial standstill with absent P waves and idioventricular rhythm.

Before his admission to hospital the patient had attended numerous doctors for this and for other illnesses. Unfortunately, in spite of diligent enquiry, no history of his having been given sulphonamides or sulphonamide derivatives could be obtained.

DISCUSSION

Many examples of renal complications from the use of sulphonamides and sulphonamide derivatives such as acetazolamide, carbutamide, and tolbutamide have been recorded. Murphy *et al.* (1944) divided the effects into obstructive lesions in the kidneys, pelvis, or ureters, and toxic effects on the kidney parenchyma. The toxic effects they subdivided into simple tubular degeneration, necrotic tubular degeneration, and glomerular changes. In kidney sections, birefringent crystals of typical appearance are found in the tubules, and interstitial inflammatory changes are frequent. The lesions are acute: chronic changes with fibrosis and contraction of the kidneys, such as were seen in the patient here described, appear to be unusual.

Oliguria or anuria with nitrogen retention is common, but severe electrolyte disturbances have not been recorded. The urine is usually normal or may contain a few red blood cells. Most of the cases reported have had acute lesions but in the one described here the history suggested a chronic

disorder of one year's duration and this was confirmed by the contracted, fibrotic kidneys seen at autopsy. Unfortunately it proved impossible to discover when the drug was administered.

Significant hyperkalæmia is unusual in chronic uræmia in the absence of increased tissue breakdown or the administration of potassium salts (Black, 1957). The patient described had been losing weight and this may have contributed to the high serum potassium level.

The electrocardiographic changes of hyperkalæmia are mainly dependent on the extracellular concentration of potassium, although this does not necessarily reflect the total body content of this ion. Bellet (1955) has described the cardiographic changes in the dog, which follow much the same pattern as in man. An increase in amplitude of the T waves followed by the development of sinus pauses are the earliest changes. Later, when the serum level of potassium is higher, the ventricular complexes widen and the ventricular rate tends to slow, although it may occasionally be rapid. At very high levels further widening of the ventricular complexes occurs and slow idioventricular rhythm develops. Death is usually due to ventricular standstill, but occasionally to ventricular fibrillation.

The early stages may be difficult to detect because the normal range of the height of T waves is wide, with an upper limit of 13 mm.; and although these normal high T waves and the high T waves of posterior infarction and of pericarditis tend to have a wider base, this is unreliable as a means of differentiation (Goodwin, 1958). When T waves are inverted prior to hyperkalæmia, they tend to become deeply inverted if the inversion is due to infarction and less deeply inverted, and finally upright, if it is due to ventricular hypertrophy (Bellet, 1955). High potassium levels have been shown on occasion to produce patterns like those of myocardial infarction (Levine *et al.*, 1956): in one of their cases cardiographic improvement followed dialysis, and autopsy later showed that no infarction was present. Low calcium and low sodium levels may enhance the toxic effects of potassium on the cardiogram (Bellet, 1955), and this may explain why in the case described such severe effects were seen at the relatively modest serum potassium level of 8.2 m.eq./l.

Summary

A patient is described who developed a chronic nephritic lesion with tubular degeneration and gross interstitial fibrosis due to sulphonamide-like crystalline deposits in the kidneys. This led to uræmia with hyperkalæmia. The hyperkalæmia gave rise to an unusual arrhythmia of repetitive runs of atrial standstill and idioventricular rhythm alternating with normal rhythm.

I should like to record my gratitude to Professor J. N. P. Davies of the Department of Pathology, Makerere, for his report on the autopsy specimens.

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